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Handbook of SYNCOPE

A Concise Clinical Approach

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Foreword
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CHAPTER 4

Additional Tests used in the Evaluation of the Syncopal Patient

INTRODUCTION

After a thorough history, a complete physical examination, and electrocardiography (ECG), there will a significant minority of patients in whom additional testing is required. It must be emphasized that if the initial evaluation is clearly diagnostic, additional "confirmatory" testing is usually unnecessary. When appropriate, head-up tilt table testing, exercise stress testing, and comprehensive electrophysiology study may be considered.[1]

HEAD-UP TILT TABLE TESTING

Although head up tilt was used in the 1950's as a tool to investigate human pathophysiology, it was not until the 1980's and 1990's that it came into widespread use as a clinical test for the diagnosis of neurocardiogenic syncopal syndromes. The test seeks to reproduce the gravitational (orthostatic) challenge associated with man's vertical posture in a controlled and monitored environment, so as to reproduce the syndrome of vasovagal syncope. Extensive literature exists on this subject, and significant insight into the pathophysiology of vasovagal syncope and related syndromes has been gained. However, the clinical role of this test remains limited in large measure because of its relatively modest sensitivity and specificity. Furthermore, the reported methodology of the test is variable: the degree of tilt (40^0–80^0, most commonly 60–70^0), the type of support (knee and torso straps, saddle support), the duration of tilting (15–60 minutes), and the type and dose of provocative agent used if the baseline test is negative (isoproterenol, sublingual nitroglycerin, etc.) are poorly standardized, therefore, making comparison of results difficult. In general, steeper angles of tilt (80^0), higher doses of isoproterenol (>1 μg/min), and prolonged duration (>10–15 min) are associated with higher numbers of false positive results.[2] The test also has poor day-to-day reproducibility and high false positive (~15%) and false negative rates (~40%). Therefore, as stated in the 2006 American Heart Association/American College of Cardiology Guidelines,[1] "in a patient of any age with an otherwise normal evaluation who has a negative tilt table test, the most likely diagnosis is still neurocardiogenic syncope". A recent meta-analysis suggested that tilt testing has an acceptable ability to discriminate between symptomatic patients and asymptomatic controls; the area under the receiver-operating characteristics curve was 0.84 and an adjusted diagnostic odds ratio was 12.15 ($p < 0.001$).[3] The results of tilt table testing are, in general, not affected by the phase of the menstrual cycle in female

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fainters, although the duration of loss of consciousness may be longer during menstruation.[4] In children, when the history and presentation do not allow accurate distinction between syncope and seizure, recent data suggest that performing video-electroencephalography (EEG) monitoring during head-up tilt testing can be helpful.[5]

When the clinical presentation of a syncopal patient is either typical of, or entirely inconsistent with, classic neurocardiogenic syncope, a tilt test is unnecessary and no further testing is required. It is only when the clinical presentation is suggestive but not typical for this diagnosis, that head-up tilt testing may be clinically useful. If decided upon, it is essential to decide *a priori* how the results will be interpreted. The following scenarios should be discussed with the patient ahead of time:

1. A negative (normal) test does not exclude the diagnosis of neurocardiogenic syncope, and is not helpful.
2. A positive test (i.e., syncope is provoked) but without reproduction of the clinical symptoms may be a false positive result, and would require careful interpretation.
3. A positive test with reproduction of the clinical syndrome is required to confirm the suspected diagnosis of neurocardiogenic syncope.
4. Repeating the test is not particularly helpful.[6] In keeping with this observation, using serial tilt testing to assess efficacy of drug therapy is also controversial, although widely reported. Treatment with placebo results in conversion of a positive tilt to a negative result in many (50–70%) patients.[7]
5. The degree of bradycardia and the length of an asystolic pause (if provoked) during head-up tilt testing is not predictive of the severity of the problem, and does not automatically imply need for permanent pacemaker implantation.[8]

EXERCISE STRESS TESTING

Syncope that occurs in the midst of exertion is the classic scenario where exercise stress testing would be of diagnostic help. In general, exertional syncope should be considered more ominous, especially if abrupt, as compared to syncope that occurs unrelated to exertion; neurocardiogenic syncope is uncommon during exertion, although it may be seen following cessation of activity. The astute clinician will always remember to obtain a detailed family history in patients with exertional syncope, since conditions, such as hypertrophic cardiomyopathy, long QT syndrome (usually LQT1), arrhythmogenic right ventricular cardiomyopathy (ARVC), and catecholaminergic polymorphic ventricular tachycardia (CPVT) are all familial traits. In this regard, once one of these familial conditions is established, it is the responsibility of the clinician to recommend familial screening for all first degree relatives. Other, nonarrhythmic causes of exertional syncope include anomalous coronary arteries and rarely, mitral valve prolapse.

During stress testing, particular attention should be paid to:

1. The behavior of the QT interval, since many patients with LQTS will show normal QT intervals on their resting ECGs, but the QT interval will either fail to shorten, or actually prolong with exercise or during recovery from exercise.[9]

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2. The burden of ventricular ectopy, since benign ventricular premature depolarizations (VPDs) typically diminish in frequency as sinus rates accelerate with exercise; however, in patients with ARVC and with CPVT, VPD burden will increase as exercise progresses, and may culminate in monomorphic (ARVC), bidirectional (CPVT), or polymorphic VT.
3. Development of atrioventricular (AV) conduction disturbances, since any worsening of AV conduction during exercise is always pathologic. Abrupt transitions from 1:1 AV conduction to 2:1 AV block will result in abrupt halving of the ventricular rate at peak exercise, often with hemodynamic compromise (see Figures 1 and 6 in chapter 8).
4. In patients with aortic stenosis and syncope, carefully monitored exercise testing, especially echocardiographic stress testing, may be of great clinical value, and may provide the correct diagnosis and treatment strategy. Any sort of stress testing in the setting of severe aortic stenosis should be undertaken with great caution, since catastrophic complications may occur.[10] Development of ischemia, ventricular arrhythmias, frank syncope, or lack of expected rise in blood pressure or a hypotensive response predict an adverse outcome, and should prompt consideration of aortic valve surgery.

COMPREHENSIVE ELECTROPHYSIOLOGY STUDY

An electrophysiology (EP) study is performed by inserting multielectrode catheters (usually via the femoral veins) under fluoroscopic guidance into the heart in order to assess sinus node, atrial, AV node, His-Purkinje, and ventricular electrical properties. The test has a very low yield (~5%) in patients with frequently recurring syncope and in patients with a structurally normal heart (as determined by examination, ECG and echocardiography). Even when a bradyarrhythmia is electrocardiographically documented as the cause of syncope, EP testing has a low yield.[11]

However, the EP study is considerably more helpful in patients with structural heart disease, and is clinically indicated in this setting. The presence of structural heart disease and nonsustained ventricular tachycardia by Holter monitoring are highly sensitive for provocation of serious ventricular tachyarrhythmias at electrophysiologic study, whereas sinus bradycardia, first-degree heart block or bundle branch block by electrocardiogram are modestly sensitive for bradyarrhythmic outcomes.[12] Hence, EP testing is less sensitive for the recognition of sinus node dysfunction as a cause of syncope (which is truly a clinical diagnosis), but has greater utility in assessing AV block and tachyarrhythmias [both supraventricular VT (SVT) and VT]. In the setting of an abnormal ECG with bundle branch block or PR interval prolongation, the likelihood of finding AV block is increased.[13] In the setting of mild LV dysfunction (EF between 40 and 50%) and known coronary artery disease, the EP study has a low likelihood of inducing VT but is still recommended. A negative (normal) EP study in patients with coronary artery disease does not always predict good long-term outcomes;[14] such patients remain at risk risk of recurrent syncope. In patients with more severe left ventricular (LV) dysfunction [ejection fraction (EF) <35–40%], and with pathologic Q waves seen on the ECG, the likelihood of inducing VT at EP testing is greater; however, current guidelines favor implantable

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cardioverter defibrillator implantation in patients with LVEF <35% irrespective of syncope, making a diagnostic EP study less important in such patients. Some important findings at EP testing that are likely to be the etiology of abrupt syncope include abnormally prolonged sinus node recovery times, prolonged HV interval, infra-His block, "split" His bundle signals (or intra-His block), and induction of rapid SVT or VT (fascicular/idiopathic VT, myocardial/scar VT or bundle branch reentrant VT). Although often performed when arrhythmic syncope is suspected based on history, EP studies in patients with a structurally normal heart and a normal or borderline ECG are usually negative (normal), even when subsequent implantable loop recorder confirms the diagnosis of paroxysmal AV block.

ROLE OF NEUROLOGIC TESTING AND ELECTROENCEPHALOGRAMS

The majority of syncope is NOT due to neurologic causes, and routine head computed tomography scans, and EEG testing show zero or extremely low diagnostic yield.[15,16,17] Only when the clinical presentation suggests a neurologic cause, a neurology consultation is appropriate.

SUMMARY POINTS

- Judicious use of head-up tilt table testing, with expectations discussed *a priori* with patients, may be of help in selected patients; the test is known to have a high rate of false positive and false negative results; not appropriate when history clearly makes vasovagal syncope unlikely
- Exercise stress testing is useful when syncope occurs during exertion; assessment of ECG changes include development of ectopy, QT interval behavior, ST segment shifts, and atrioventricular conduction disturbances; avoid stress testing in setting of aortic stenosis
- Comprehensive EP study is unhelpful in patients with normal ECGs and absence of any structural heart disease (diagnostic yield in this type of patient is <5%); EP testing is more helpful in patients with syncope that is concerning for arrhythmic cause, when ECG is abnormal, and when structural heart disease is present.

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About the author

Dr Behzad Pavri has spent his entire medical career in academic medicine. After graduating from University of Bombay, Seth G. S. Medical College, Dr Pavri moved to the US in 1986. After Internal Medicine residency training at Harlem Hospital Center in New York, Dr Pavri went on to fellowships in Cardiovascular Diseases at Columbia-Presbyterian Medical Center, Columbia University in New York, and in Cardiac Electrophysiology at Massachusetts General Hospital, Harvard University in Boston. He was on Faculty at the Hospital of the University of Pennsylvania from 1994 through 2001, and has since been on faculty at Thomas Jefferson University Hospital in Philadelphia, where he is Professor of Medicine and the Director of the Cardiac Electrophysiology Fellowship Program.

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